Austin Spine

Open Access

Austin Publishing Group

Review Article

Perioperative Pulmonary Embolism Prevention and Treatment

Huang J[1]*, Lu Y[2] and Chen J[1]

[1]Department of Medicine, University of Central Florida, USA

[2]Department of Medicine, Hennepin County Medical Center, USA

***Corresponding author:** Huang J, Department of Medicine, University of Central Florida, Orlando, FL, USA

Received: January 05, 2017; **Accepted:** February 02, 2017; **Published:** February 06, 2017

Abstract

All perioperative patients are at an increased risk of pulmonary embolism and venous thromboembolism. Perioperative massive Pulmonary Embolism (PE) is a significant cause of morbidity and mortality. Clinical outcomes have been shown to be improved by a high index of suspicion, prompt recognition, and aggressive intervention. It is important that healthcare providers recognize perioperative PE and know prevention and treatment options. Many medical societies have published guideline recommendations for management of PE. In this review, we will focus on perioperative acute PE treatment and prevention to implement guideline recommendations for optimizing management of acute PE.

Keywords: Pulmonary Embolism; Diagnosis; Thrombolytic agents; Prevention

Introduction

Massive perioperative Pulmonary Embolism (PE) is an uncommon event but significant cause of morbidity and mortality. It is estimated that PE is responsible for between 150,000 and 200,000 deaths per year in the United States [1]. 30% of the deaths from PE take place during the perioperative period [1]. PE is the third most common cardiovascular disease after myocardial infarction and cerebrovascular accident (stroke). Several studies have reported mortality rates ranging from 15% to 30%, while mortality rates in a massive PE can reach 30% to 50% [2-4]. A recent review of more than 3000 massive intraoperative thromboembolic events revealed an overall mortality of 41% [5].

Surgery increases the risks for perioperative PE. Healthcare providers, including anesthesiologists, are responsible for the diagnosis and treatment of perioperative PE. During surgery, PE often first presents with hemodynamic instability and if progressing quickly, can lead to death. It is important that healthcare providers recognize perioperative PE and know prevention and treatment options. Prompt diagnosis and treatment can save patient lives. In this review, we will focus on perioperative acute PE treatment and prevention.

Diagnosis of PE

Diagnosis of a PE in the perioperative period can be a challenge, but early detection can reduce morbidity. The American Heart Association (AHA) classified and defined PE into three classes: massive PE, submassive PE, and low-risk PE [6].

Definition for massive PE: Acute PE with sustained hypotension (systolic blood pressure <90 mm Hg for at least 15 minutes or requiring isotropic support, not due to a cause other than PE, such as arrhythmia, hypovolemia, sepsis, or Left Ventricular (LV) dysfunction), pulselessness, or persistent profound bradycardia (heart rate<40 bpm with signs or symptoms of shock) [6].

Definition for submassive PE: Acute PE without systemic hypotension (systolic blood pressure ≥ 90 mm Hg) but with either RV dysfunction or myocardial necrosis [6].

RV dysfunction means the presence of at least 1 of the following: (1) RV dilation (apical 4-chamber RV diameter divided by LV diameter >0.9) or RV systolic dysfunction on echocardiography. (2) RV dilation (4-chamber RV diameter divided by LV diameter >0.9) on CT. (3) Elevation of BNP (>90 pg/mL). (4) Elevation of N-terminal pro-BNP (>500 pg/mL); or (5) Electrocardiographic changes (new complete or incomplete right bundle-branch block

ST elevation or depression, or anteroseptal T-wave inversion).

Myocardial necrosis is defined as either of the following: (1) Elevation of troponin I (>0.4 ng/mL) or (2) Elevation of troponin T (>0.1 ng/mL).

Definition for low-risk PE: Acute PE and the absence of the clinical markers of adverse prognosis that define massive or submassive PE [6].

The Treatment of Acute Pulmonary Embolism

Systemic thrombolysis

Thrombolytic agents are indicated in patients who are normotensive but with evidence of RV failure or in cases of hemodynamic instability [7]. Several societies guidelines advocate the use of thrombolytic agent in patients with hemodynamic compromise and massive PE is acceptable [6,8].

A meta-analysis of clinical trials including patients with massive PE showed IV fibrinolytic agents reduced the composite of recurrent PE and death, but not in death alone [9]. The results of patients with submassive PE were better characterized in randomized trials. These studies showed the use of IV fibrinolytic therapy in patients with massive or submassive PE can improve hemodynamic stability and, possibly reduce the risk of recurrent PE and PE-attributed death [10].

The most commonly used thrombolytic agents approved by the

Citation: Huang J, Lu Y and Chen J. Perioperative Pulmonary Embolism Prevention and Treatment. Austin Spine. 2017; 1(1): 1001.

Table 1: AHA recommendations for systemic thrombolysis for acute PE [6].

Recommendations	Class	Level
Fibrinolysis is reasonable for patients with massive acute PE and acceptable risk of bleeding complications	IIa	B
Fibrinolysis may be considered for patients with submassive acute PE judged to have clinical evidence of adverse prognosis (new hemodynamic instability, worsening respiratory insufficiency, severe RV dysfunction, or major myocardial necrosis) and low risk of bleeding complications	IIb	C
Fibrinolysis is not recommended for patients with low-risk PE or submassive acute PE with minor RV dysfunction, minor myocardial necrosis, and no clinical worsening	III	B
Fibrinolysis is not recommended for undifferentiated cardiac arrest	III	B

Recommendation class: I: benefits>>>risks, IIa: benefits>>risks, IIb: benefits ≥ risks; III: risks ≥ benefits.
Level of evidence: A: Multiple population evaluated; B: Limited population evaluated; C: Very limited population evaluated.

Table 2: AHA recommendation for s catheter based therapies and surgical embolectomy for acute PE [6].

Recommendations	Class	Level
Depending on local expertise, either catheter embolectomy and fragmentation or surgical embolectomy is reasonable for patients with massive PE and contraindications to fibrinolysis	IIa	C
Catheter embolectomy and fragmentation or surgical embolectomy is reasonable for patients with massive PE who remain unstable after receiving fibrinolysis	IIa	C
For patients with massive PE who cannot receive fibrinolysis or who remain unstable after fibrinolysis, it is reasonable to consider transfer to an institution experienced in either catheter embolectomy or surgical embolectomy if these procedures are not available locally and safe transfer can be achieved	IIa	C
Either catheter embolectomy or surgical embolectomy may be considered for patients with submassive acute PE judged to have clinical evidence of adverse prognosis (new hemodynamic instability, worsening respiratory failure, severe RV dysfunction, or major myocardial necrosis)	IIb	C
Catheter embolectomy and surgical thrombectomy are not recommended for patients with low-risk PE or submassive acute PE with minor RV dysfunction, minor myocardial necrosis, and no clinical worsening	III	C

Table 3: AHA recommendation for placement of IVC filter for acute PE [6].

Recommendations	Class	Level
Adult patients with any confirmed acute PE (or proximal DVT) with contraindications to anticoagulation or with active bleeding complication should receive an IVC filter	I	B
Anticoagulation should be resumed in patients with an IVC filter once contraindications to anticoagulation or active bleeding complications have resolved	I	B
Patients who receive retrievable IVC filters should be evaluated periodically for filter retrieval within the specific filter's retrieval window	I	C
For patients with recurrent acute PE despite therapeutic anticoagulation, it is reasonable to place an IVC filter	IIa	C
For DVT or PE patients who will require permanent IVC filtration (e.g., those with a long-term contraindication to anticoagulation), it is reasonable to select a permanent IVC filter device	IIa	C
For DVT or PE patients who will require permanent IVC filtration (e.g., those with a long-term contraindication to anticoagulation), it is reasonable to select a permanent IVC filter device	IIa	C
Placement of an IVC filter may be considered for patients with acute PE and very poor cardiopulmonary reserve, including those with massive PE	IIb	C
An IVC filter should not be used routinely as an adjuvant to anticoagulation and systemic fibrinolysis in the treatment of acute PE	III	C

US Food and Drug Administration (FDA) for acute PE included: Recombinant tissue type Plasminogen Activator (tPA, alteplase), Streptokinase (SK) and recombinant human Urokinase (UK). Other thrombolytic agents not FDA approved include tenecteplase and reteplase.

All fibrinolytic drugs are enzymes that convert the patient's native circulating plasminogen into plasmin. The contraindications include active internal bleeding; prior intracranial hemorrhage, intracranial cerebrovascular disease, suspected aortic dissection, intracranial malignant neoplasm, ischemic stroke within 3 months, recent intracranial or spinal cord surgery, recent closed-head or facial trauma with fracture or intracerebral injury [6], and severe uncontrolled hypertension [11].

Streptokinase should also not be used after 5 days to 12 months of initial use for possible anaphylactic reaction from anti-streptokinase antibodies or in patients with recent streptococcal infections due to possible drug resistance or reduced effects (Table 1) [12-15] .

Catheter-based therapies

Catheter-based therapies can (1) rapidly reduce pulmonary artery pressure, RV strain, and Pulmonary Vascular Resistance (PVR), (2) increase systemic perfusion and (3) facilitate RV recovery [6]. This treatment is an alternative method to remove pulmonary emboli and is a less invasive approach compared to surgical embolectomy. Catheter-directed therapies include mechanical fragmentation of thrombus with a standard pulmonary artery catheter, clot pulverization with a rotating basket catheter, percutaneous rheolytic thrombectomy, or pigtail rotational catheter embolectomy [16]. Catheter-directed thrombolysis is considered in cases of unsuccessful systemic thrombolysis, contraindications to thrombolytic therapy, and when surgical embolectomy is unavailable or not feasible (Table 2) [7].

Potential complications from catheter directed therapies include pulmonary hemorrhage and right atrial or ventricular perforation leading to cardiac tamponade. Perforation or dissection of a major pulmonary artery branch may cause acute massive pulmonary hemorrhage and death [6].

There are no randomized trialsor prospective cohort studies that have evaluated catheter based techniques for massive PE [16]. A systematic review of available cohort data included a total of 348

Table 4: Modified Caprini assessment model for general surgery thrombotic risk evaluation.

1 Point	2 Points	3 Points	5 Points
Age 41-60 y	Age 61-74 y	Age ≥75 y	Stroke (<1 mo)
Minor surgery	Arthroscopic surgery	History of VTE	Elective arthroplasty
BMI >25 kg/m^2	Major open surgery (>45 min)	Family history of VTE	Hip, pelvic, or leg fracture
Swollen legs	Laparoscopic surgery (>45 min)	Factor V Leiden	Acute spinal cord injury (<1 mo)
Varicose veins	Malignancy	Prothrombin 20210A	
Pregnancy or postpartum	Confined to bed (>72 h)	Lupus anticoagulation	
History of unexplained or recurrent spontaneous abortion	Immobilizing plaster cast	Anticardiolipin antibodies	
Oral contraceptive or hormone replacement	Central venous access	Elective serum homocysteine	
Sepsis (<1 mo)		Heparin-induced thrombocytopenia	
Serious lung disease, including pneumonia (<1 mo)		Other congenital or acquired thrombophilia	
Abnormal pulmonary function			
Acute myocardial infarction			
Congestive heart failure (<1 mo)			
History of inflammatory bowel disease			
Medical patient at bed rest			

patients [17]. Clinical success with percutaneous therapy alone for patients with acute massive PE was 81%, and clinical success when combined with local infusion of thrombolytic agents was 95% [17].

Surgical embolectomy

Surgical embolectomy is considered the last treatment option for acute PE [18]. The surgery requires a median sternotomy and cardiopulmonary bypass. This operation is indicated for acute PE patients who need surgical excision of a right atrial thrombus, impending paradoxical arterial embolism, or a closure of a patent foramen ovale [16]. Surgical embolectomy can be used for patients in whom thrombolysis has been unsuccessful [16]. Significant advances in cardiac surgical techniques have reduced surgical mortality, which is about 6% currently [19,20]. In addition, there is evidence that pulmonary embolectomy can reduce long term mortality [21,22].

Major risks of surgical embolectomy include: injury to the distal branches of the PA during embolectomy that can lead to significant bronchoalveolar hemorrhage [7], inability to wean from cardiopulmonary bypass because of primary RV dysfunction, persistent severe pulmonary hypertension, or severe hypoxia that could require the use of mechanical circulatory support/ Extracorporeal Membrane Oxygenation (ECMO) as a bridge to recovery [7].

Vena caval filters

Placement of an Inferior Vena Cava (IVC) filter is indicated in patients with acute PE who have absolute contraindications to anticoagulation, in those experiencing major bleeding events during the acute phase, and in patients with objectively confirmed recurrent PE, despite adequate anticoagulation treatment [23].

Data from the US Nationwide Inpatient Sample suggest that cava filters may be associated with an improved outcome [24]. In the PREPIC Trial (Pre´vention du Risqued' Embolie Pulmonairepar Interruption Cave), hospitalized patients with acute symptomatic PE associated with lower-limb vein thrombosis were randomized into two groups, one group received anticoagulation only, the other group received anticoagulation with IVC filter [25]. The results showed that there were no differences in major bleeding, post thrombotic chronic venous insufficiency, or death during the study period [25]. These results did not demonstrate any advantages of the use of retrievable IVC filters in patients with acute PE.

Potential complications of IVC included penetration of the caval wall or embolization to the right heart cavities (Table 3).

Prevention

Deep Vein Thrombosis (DVT) occurred in 348, 558 hospitalizations, Pulmonary Embolism (PE) occurred in 277, 549 hospitalizations, and concomitant DVT and PE occurred in 78, 511 hospitalizations each year [26]. It was estimated that 15 percent, 24 percent, and 17 percent were at moderate, high, or very high risk for venous thromboembolism (VTE includes both deep vein thrombosis and pulmonary embolism) [27].

The National Quality Forum, the Surgical Care Improvement Project, the Centers for Medicine and Medicinal Services, the Joint Commission on Accreditation of Health Care Organizations, and the Office of the Surgeon General of the United States all have initiatives for VTE prophylaxis.

The American College of Chest Physicians (ACCP) published a series of VTE guidelines, and when comparing it with the previous guidelines, a very important change in the ACCP 2012 guideline is the emphasis in individualized assessment [28-30]. VTE perioperative evaluation should include the type and extent of surgery or trauma, duration of hospitalization, a history of previous VTE or cancer, immobility, recent sepsis, presence of a central venous access device, pregnancy or the postpartum period, and inherited or acquired hypercoagulable states. All clinical decisions should be made based on the balance between the risk of VTE and risk of major bleeding in the consideration of available literature reports (Table 4).

Perioperative patients can be divided in to four different risks categories of VTE, patient with high risks, moderate risks, low risks and very low risks [28-30].

High risk patients -Patients undergoing general and abdominal-pelvic surgery with a Caprini score of 5 or more, or those undergoing plastic and reconstructive surgery with a Caprini score of 7 to 8 are considered as high risk patients [29-31]. The estimated baseline risk of VTE in the absence of prophylaxis is estimated to be approximately 6 percent. Examples of patients in the high risk group are those undergoing hip/knee arthroplasty, pelvic/hip fracture surgery, colorectal surgery, major trauma, spinal cord injury or cancer surgery [28-30].

The VTE prophylaxis protocol for patients with high risk patients, recommends the use of either drugs or physical methods that are effective for preventing DVT and is considered as a primary prevention approach. Secondary prevention involves the early detection and treatment of subclinical venous thrombosis. Primary prophylaxis is preferred and is more cost effective than treatment after a VTE [27].

With all primary VTE prevention in patients with high risks without major bleeding risk, pharmacology prevention is preferred [30,31]. These agents include low-molecular-weight heparin; fondaparinux; dabigatran, apixaban, rivaroxaban, endoxaban; low-dose unfractionated heparin; adjusted-dose vitamin K antagonist; aspirin (all Grade I B) for a minimum of 10 to 14 days [28].

Patients at high risk for VTE undergoing abdominal or pelvic surgery for cancer, ACCP recommends extended-duration, postoperative, pharmacologic prophylaxis for 4 weeks with LMWH over limited-duration prophylaxis (Grade IB) [30].

Patients with high risk of VTE undergoing orthopedic surgery are suggested to be on VTE prophylaxis for up to 35 days (Grade II B) [28]. In patients at increased bleeding risk, ACCP suggests an IPCD or no prophylaxis [28].

For patients with isolated lower extremity injuries requiring leg immobilization, ACCP suggests no thromboprophylaxis (Grade IIB). For patients undergoing knee arthroscopy without a history of VTE, no thromboprophylaxis is suggested either (Grade IIB) [28].

Patients with a high risk for VTE who are at high risk for major bleeding complications or those in whom the consequences of bleeding are believed to be particularly severe, ACCP the recommends the use of mechanical prophylaxis, preferably with IPC, over no prophylaxis until the risk of bleeding diminishes and pharmacologic prophylaxis may be initiated (Grade IIC) [30].

Moderate risk patients -Surgical patients undergoing general and abdominal-pelvic surgery with a Caprini score of 3 to 4, or those undergoing plastic and reconstructive surgery with a Caprini score of 5 to 6 carry a moderate risk of thrombotic events. Their estimated baseline risk of VTE in the absence of prophylaxis is estimated to be approximately 3 percent. Examples of these groups of patients include patients with general gynecologic, urologic, thoracic, ankle fracture, or neurosurgical procedures [28-30].

For patient with moderate risk of VTE without major risk of bleeding: Low-Molecular-Weight Heparin (LMWH) (Grade IIB), low-dose unfractionated heparin (Grade IIB), or mechanical prophylaxis with IPC (Grade IIC) is recommended over no prophylaxis [30]. For patients at moderate risk for VTE who are at high risk for major bleeding complications or those in whom the risk of bleeding is severe, the ACCP recommends mechanical prophylaxis, preferably with IPC until the risk of bleeding diminishes and pharmacologic prophylaxis may be initiated (Grade IIC) [31].

Low risk patients - Patients undergoing general and abdominal-pelvic surgery with a Caprini score of 1 to 2, or those undergoing plastic and reconstructive surgery with a Caprini score of 3 to 4 are included in the low risk group for thrombotic events. Their estimated baseline risk of VTE in the absence of prophylaxis is estimated to be approximately 1.5 percent. Clinical data on this group is scarce but the recommendation by the ACCP includes mechanical prophylaxis, preferably with intermittent pneumatic compression (IPC) over no prophylaxis (Grade IIC) [30].

Very low risk patients - patients undergoing general and abdominal-pelvic surgery with a Caprini score of zero, and those undergoing plastic and reconstructive surgery with a Caprini score of zero to two carries very low risk of thrombotic events with estimated risk less than 0.5 percent without prophylaxis. There is no clinical data to demonstrate the efficacy of VTE prophylaxis in this group. No specific pharmacologic (Grade I B) or mechanical (Grade II C) prophylaxis are recommended to be used other than early ambulation [30].

An important update in the 2012 ACCP guideline is for patients in all risk groups, that recommend that an inferior vena cava filter should not be used for primary VTE prevention (Grade II C) and that surveillance with venous compression ultrasonography should not be performed (Grade II C) [28-30].

VTE Prophylaxis should be started either before or shortly after surgery, and continued at least until the patient is fully ambulatory based on FDA approved labeling.

References

1. Galson SK. The surgeon general's call to action to prevent deep vein thrombosis and pulmonary embolism.
2. Brembilla-Perrot B, Miljoen H, Houriez P, Beurrier D, Nippert M, Vançon AC, et al. Causes and prognosis of cardiac arrest in a population admitted to a general hospital; a diagnostic and therapeutic problem. Resuscitation. 2003; 58: 319-327.
3. Torbicki A, Galié N, Covezzoli A, Rossi E, De Rosa M, Goldhaber SZ, et al. Right heart thrombi in pulmonary embolism. Results from the International Cooperative Pulmonary Embolism Registry. J Am Coll Cardiol. 2003; 41: 2245-2251.
4. Dalen JE, Alpert JS. Natural history of pulmonary embolism. Prog Cardiovasc Dis. 1975; 17: 259-270.
5. Visnjevac O, Lee K, Bulatovic R, Pourafkari L, Porhomayon J, Nader ND. Outcomes based systematic review for management of massive intra-cardiac or pulmonary thrombotic emboli during surgery. Heart Lung Vessel. 2014; 6: 24-32.
6. Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, Goldhaber SZ, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. Circulation. 2011; 123: 1788-1830.

7. Cox JC, Jablons DM. Operative and Perioperative Pulmonary Emboli. Thorac Surg Clin. 2015; 25: 289-299.

8. Torbicki A, Perrier A, Konstantinides S, Agnelli G, Galie N, Pruszczyk P, et al. Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). Eur Heart J. 2008; 29: 2276-2315.

9. Becattini C, Vedovati MC, Agnelli G. Prognostic value of troponins in acute pulmonary embolism. A meta-analysis. Circulation. 2007; 116: 427-433.

10. Chatterjee S, Chakraborty A, Weinberg I, Kadakia M, Wilensky RL, Sardar P, et al. Thrombolysis for pulmonary embolism and risk of all-cause mortality, major bleeding, and intracranial hemorrhage: a meta-analysis. JAMA. 2014; 311: 2414-2421.

11. O'Gara PT, Kushner FG, Ascheim DD, Casey DE, Chung MK, de Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013; 127: 529-555.

12. McGrath KG, Patterson R. Anaphylactic reactivity to streptokinase. JAMA. 1984; 252: 1314-1317.

13. Tio RA, Voorbij RH, Enthoven R. Adult respiratory distress syndrome after streptokinase. Am J Cardiol. 1992; 70: 1632-1633.

14. Dykewicz MS, Patterson R. Serum sickness from streptokinase. Am Heart J. 1988; 115: 1338-1339.

15. Noel J, Rosenbaum LH, Gangadharan V, Stewart J, Galens G. Serum sickness-like illness and leukocytoclastic vasculitis following intracoronary arterial streptokinase. Am Heart J. 1987; 113: 395-397.

16. Kearon C, Kahn SR, Agnelli G, Goldhaber S, Raskob GE, Comerota AJ. Antithrombotic therapy for venous thromboembolic disease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). CHEST. 2008; 133: 454S-545S.

17. Skaf E, Beemath A, Siddiqui T, Janjua M, Patel NR, Stein PD. Catheter-tip embolectomy in the management of acute massive pulmonary embolism. Am J Cardiol. 2007; 99: 415-420.

18. Jaber WA, Fong PP, Weisz G, Lattouf O, Jenkins J, Rosenfield K, et al. Acute Pulmonary Embolism: With an Emphasis on an Interventional Approach. J Am Coll Cardiol. 2016; 67: 991-1002.

19. Stulz P, Schläpfer R, Feer R, Habicht J, Grädel E. Decision making in the surgical treatment of massive pulmonary embolism. Eur J CardiothoracSurg. 1994; 8: 188-193.

20. Yalamanchili K, Fleisher AG, Lehrman SG, Axelrod HI, Lafaro RJ, Sarabu MR, et al. Open pulmonary embolectomy for treatment of major pulmonary embolism. Ann ThoracSurg. 2004; 77: 819-823.

21. DigonnetA, Moya-Plana A, Aubert S, Flecher E, Bonnet N, Leprince P, et al. Acute pulmonary embolism: a current surgical approach. Interact Cardiovasc Thorac Surg. 2007; 6: 27-29.

22. Leacche M, Unic D, Goldhaber SZ, Rawn JD, Aranki SF, Couper GS, et al. Modern surgical treatment of massive pulmonary embolism: results in 47 consecutive patients after rapid diagnosis and aggressive surgical approach. J Thorac Cardiovasc Surg. 2005; 129: 1018-1023.

23. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galiè N, et al. 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014; 35: 3033-3069.

24. Stein PD, Matta F, Keyes DC, Willyerd GL. Impact of vena cava filters on in-hospital case fatality rate from pulmonary embolism. Am J Med. 2012; 125: 478-484.

25. Decousus H, Leizorovicz A, Parent F, Page Y, Tardy B, Girard P, et al. A clinical trial of vena caval filters in the prevention of pulmonary embolism in patients with proximal deep-vein thrombosis. Pre´ventiondu Risqued' Embolie Pulmonaire par Interruption Cave Study Group. N Engl J Med. 1998; 338: 409-416.

26. Ho KM, Bham E, Pavey W Incidence of Venous Thromboembolism and Benefits and Risks of Thromboprophylaxis After Cardiac Surgery: A Systematic Review and Meta-Analysis. J Am Heart Assoc. 2015; 4: e002652.

27. Anderson FA, Zayaruzny M, Heit JA, Fidan D, Cohen AT. Estimated annual numbers of US acute-care hospital patients at risk for venous thromboembolism. Am J Hematol. 2007; 82: 777-782.

28. Falck-Ytter Y, Francis CW, Johanson NA, Curley C, Dahl OE, Schulman S, et al. Prevention of VTE in Orthopedic Surgery Patients: Chest. 2012; 141: e278S-e325S.

29. Schiff RL, Kahn SR, Shrier I, Strulovitch C, Hammouda W, Cohen E, et al. Identifying Orthopedic Patients at High Risk for Venous Thromboembolism Despite Thromboprophylaxis. Chest. 2005; 128: 3364-3371.

30. Gould MK, Garcia DA, Wren SM, Karanicolas PJ, Arcelus JI, Heit JA, et al. Prevention of VTE in nonorthopedic surgical patients. Chest. 2012; 141: e227S-e277S.

Austin Spine - Volume 1 Issue 1 - 2017
Submit your Manuscript | www.austinpublishinggroup.com

Citation: Huang J, Lu Y and Chen J. Perioperative Pulmonary Embolism Prevention and Treatment. Austin Spine. 2017; 1(1): 1001.